

The Case | Acute heart failure with elevated cardiac enzymes

S-D Chung^{1,2}, K-H Huang², C-H Liao^{2,3}, H-C Tai², H-J Yu² and S-C Chueh²

¹Division of Urology, Department of Surgery, Far Eastern Memorial Hospital, Ban Ciao, Taipei, Taiwan; ²Department of Urology, National Taiwan University Hospital and National Taiwan University, College of Medicine, Taipei, Taiwan and ³Division of Urology, Department of Surgery, Cardinal Tien Hospital, Taipei, Taiwan

Correspondence: S-C Chueh, Department of Urology, National Taiwan University Hospital, Room 11-09, No. 7, Chung Shan South Road, Taipei, Taiwan. E-mail: scchueh@ntu.edu.tw



Figure 1 | Chest radiograph at admission.

A 42-year-old man, with a history of hypertension, on no medications, was admitted with chest pain. Physical examination was notable for an afebrile patient, with blood pressure of 210/110 mm Hg; pulse, 84 beats/min, and chest exam revealed bilateral crackles. A chest radiograph (Figure 1) showed cardiomegaly and pulmonary edema. Laboratory tests showed serum B-type natriuretic peptide level 2477 pg/ml (<100 pg/ml), serum creatinine 2.3 mg/dl, creatine kinase 3769 U/l (MB fraction, 289 U/l), Troponin-I 66 ng/ml, arterial blood gas pH 7.21, PCO₂ 37.4 mm Hg, PO₂ 87.6 mm Hg, and bicarbonate 14.4 mEq/l. On the electrocardiogram, ST segment elevation was noted in leads

V1–V3; and echocardiography revealed normal ventricular size and wall thickness, with global myocardial hypokinesis. The patient was endotracheally intubated and placed on mechanical ventilation. Cardiac catheterization revealed patent coronary arteries. Subsequent to this, the patient developed shock with a blood pressure down to 70/40 mm Hg accompanied by worsening gas exchange. Intra-aortic balloon pump and then extracorporeal membrane oxygenation were utilized to provide respiratory and cardiovascular support. After aggressive cardiopulmonary support, his hemodynamics stabilized. The serum creatinine peaked at 2.8 mg/dl.

What is the differential diagnosis of this clinical picture?

SEE NEXT PAGE FOR ANSWERS

The Diagnosis | Left adrenal pheochromocytoma presenting with labile hypertension and acute heart failure

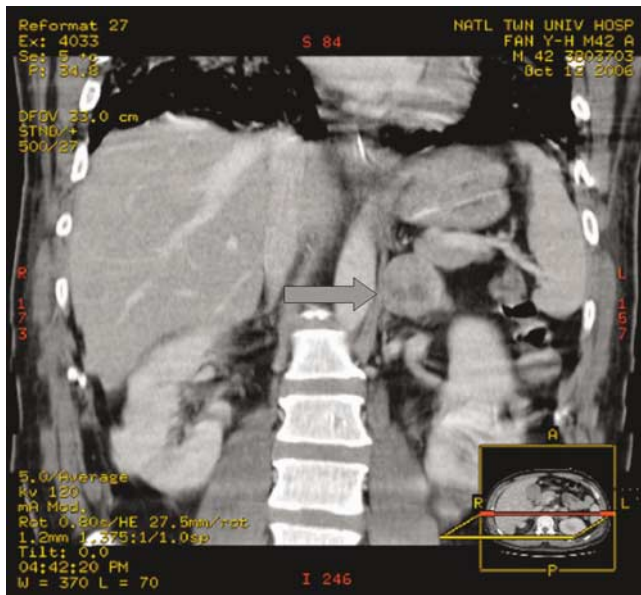


Figure 2 | Abdomen computed tomography demonstrated a left suprarenal tumor (arrow).

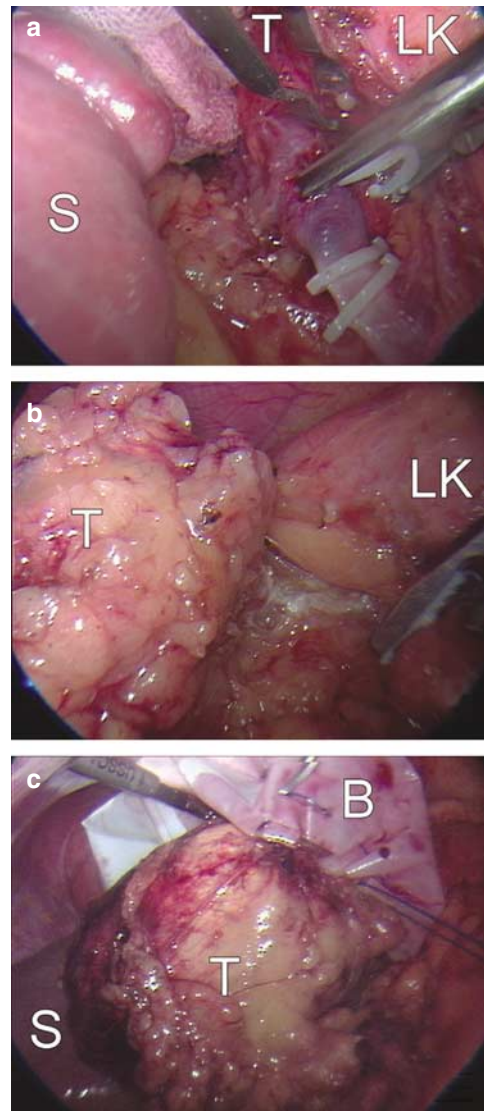


Figure 3 | Intraoperative photographs of laparoscopic adrenalectomy. (a) The left adrenal vein draining the tumor (T) being ligated with Hemo-Lok clips. S = spleen; LK = left kidney. **(b)** The plane between the tumor (T) and left kidney (LK) being dissected. **(c)** The resected tumor (T) before been put into the retrieval bag (B).

Twenty-four hours urinary vanillylmandelic acid concentration was 10.4 mg (<7 mg/24 h), epinephrine was elevated at 207.3 μ g (<22.4 μ g/24 h), and norepinephrine was 151.9 μ g (12.1–85.5 μ g/24 h). An abdomen computed tomography showed a 3.5 \times 2.7 cm heterogenous soft tissue density at

left suprarenal area (Figure 2). His respiratory and cardiac function improved gradually under intensive care. He received phenoxybenzamine to control high blood pressure and underwent laparoscopic left adrenalectomy (Figure 3); the serum creatinine improved to 0.9 mg/dl postoperatively.

After 3 months, his blood pressure was 122/82 mm Hg and he was off all antihypertensive medications.

DISCUSSION

Pheochromocytoma may present in unusual ways including dilated cardiomyopathy, pulmonary edema, cardiogenic shock, and sudden death.¹ In fact, there have been cases where the diagnosis was realized after cardiac transplantation was performed for dilated cardiomyopathy.² The classic triad of palpitation, headache, and diaphoresis may not be present (as in our case), and it is difficult to diagnose this condition in patients presenting with cardiogenic shock and acute heart failure.

De Wilde *et al.*³ described that one of the mechanisms of cardiogenic shock in patients with pheochromocytoma is hemorrhagic necrosis of the tumor, which will result in abrupt cessation of the release of catecholamine and reduced receptor activity due to previous adrenergic excess; hemorrhage in the tumor with hemosiderin deposit was also observed in the pathologic examination of the left adrenal tumor in this case. On the other hand, myocardial dysfunction due to catecholamine-related injury of myocardial fibers has also been described.⁴

To establish a diagnosis of pheochromocytoma, biochemical tests included plasma concentrations of catecholamine, metanephrine, and 24-h urine catecholamines, metanephrines, and vanillylmandelic acid output were utilized.

The diagnosis is confirmed by an abdomen computed tomography or magnetic resonance imaging. Magnetic resonance imaging is considered a more reliable and specific modality to diagnose pheochromocytomas, whose characteristic signal intensity on the T2-weighted images was characteristic for pheochromocytomas. In addition, in some rare cases of extra-adrenal or small pheochromocytomas and adrenal medullary hyperplasia, ¹³¹I-metaiodobenzylguanidine scintigraphy is quite helpful with a high specificity to localize the catecholamine-secreting lesion.⁵ In our case, the rapid diagnosis and subsequent surgical resection of the left adrenal pheochromocytoma was possible leading to a good outcome for the patient. In conclusion, a diagnosis of pheochromocytoma should be considered in patients presenting with unexplained acute heart failure.

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